MERCURY, INORGANIC

(liquid silver; hyfarargyrum; colloidal mercury)

CAS Registry Number: 7439-97-6

I. Chronic Toxicity Summary

Inhalation reference exposure level	**0.09 mg/m^3 - This REL is intended for use in assessing mercury salts as well as elemental mercury.**
Critical effects	Hand tremor, memory disturbances, neurobehavioral and autonomic dysfunction in humans
Hazard index target(s)	Nervous system

II. Chemical Property Summary (HSDB, 1995)

Description	Silvery, odorless, heavy liquid
Molecular formula	Hg
Molecular weight	200.59 g/mol
Boiling point	Hg°: 356.7 °C; $HgCl_2$: 302°C
Vapor pressure	0.002 torr @ 25°C
Solubility	Soluble in concentrated nitric and hot sulfuric acids; dissolves to some extent in lipids
Conversion factor	1 ppm = 8.2 mg/m^3

III. Major Uses or Sources

The uses of mercury and mercury containing compounds are considerable. Because it has uniform volume expansion with increasing temperature over the entire temperature range of its liquid state, it is widely used in barometers, thermometers, hydrometers, and pyrometers. It is used in mercury arc lamps producing ultraviolet rays, in fluorescent lamps, as a catalyst in oxidation of organic compounds, extracting gold and silver from ores, electric rectifiers, the making of mercury fulminate, for Millon's Reagent, and as a cathode in electrolysis. It is also used in pulp and paper manufacturing, as a component of batteries, in amalgams (dental preparations), and in the manufacture of switching devices such as oscillators, the manufacture of chlorine and caustic soda, as a lubricant, and a laboratory reagent.

To lesser extent is has been used to fumigate and protect grain from insect infestation, in pharmaceuticals, agricultural chemicals, and antifouling paints (ACGIH, 1991). The annual statewide industrial emissions in California based on the most recent inventory were estimated to be 9714 pounds of mercury (CARB, 1999).

IV. Effect of Exposure to Humans

The primary effects of chronic exposure to mercury vapor are on the central nervous system. Chronic duration exposures to elemental mercury have resulted in tremors (mild or severe), unsteady walking, irritability, poor concentration, short-term memory deficits, tremulous speech, blurred vision, performance decrements, paresthesia, and decreased nerve conduction (Albers *et al.*, 1988; Chaffin *et al*;, 1973; Fawer *et al.*, 1983; Kishi *et al.*, 1993; Langolf *et al.*, 1978; Piikivi *et al.*, 1984; Smith *et al.*, 1970). Motor system disturbance can be reversible upon cessation of exposure, however, memory deficits may be permanent (Chaffin *et al.*, 1973). Studies have shown effects such as tremor and decreased cognitive skills in workers exposed to approximately 25 $\mu g/m^3$ mercury vapor (Piikivi *et al.*, 1984; Piikivi and Hanninen, 1989; Piikivi and Toulonen, 1989) (see discussion below).

The kidney is also a sensitive target organ of mercury toxicity. Effects such as proteinuria, proximal tubular and glomular changes, albuminuria, glomerulosclerosis, and increased urinary N-acetyl-β-glucosaminidase have been seen in workers exposed to approximately 25-60 $\mu g/m^3$ mercury vapor (Barregard *et al.*, 1988; Bernard *et al.*, 1987; Roels *et al.*, 1982; Piikivi and Ruokonen, 1989).

Chronic exposure to mercury vapors has also resulted in cardiovascular effects such as increased heart and blood pressure (Fagala and Wigg, 1992; Taueg *et al.*, 1992; Piikivi, 1989) and in leukocytosis and neutrophilia (Fagala and Wigg, 1992).

A number of other studies with similar exposure levels also found adverse psychological and neurological effects in exposed versus unexposed individuals. Fawer *et al*. (1983) measured intention tremor in 26 male workers (mean age of 4 years) exposed to low concentrations of mercury vapor. The men worked either in a chloralkali plant (n = 12), a fluroescent tube manufacturing plant (n = 7), or in acetaldehyde production (n = 7) Twenty-five control subjects came from different parts of the same plants and were not occupationally exposed to mercury. The average exposure as measured by personal air sampling was 0.026 mg/m^3 and the average duration of exposure was 15 years. The measurements of intention tremor were significantly higher in exposed workers than in controls. Using the average exposure as a LOAEL and adjusting for occupational ventilation rates and workweek, the resultant LOAEL is 0.009 mg/m^3.

Piikivi and Tolonen (1989) used EEGs to study the effects of long-term exposure to mercury vapor in 41 chloralkali workers exposed for a mean of 15.6 years as compared to matched controls. They found that exposed workers who had blood mercury levels of 12 $\mu g/L$ tended to have an increased number of EEG abnormalities when analyzed by visual inspection. When analyzed by computer, brain activity was found to be significantly lower than matched controls. The changes were most prominent in the parietal cortex, but absent in the frontal cortex. The authors used a conversion factor calculated by Roels *et al*. (1987) to extrapolate from blood mercury levels of 12 $\mu g/L$ to an air concentration of 0.025 mg/m^3.

Another study by Piikivi (1989) examined subjective and objective symptoms of autonomic dysfunction in 41 chloralkali workers exposed to mercury vapor for an average of 15.6 years as compared with matched controls. The exposed workers had mean blood levels of 11.6 μg/L corresponding to a TWA exposure of 25 μg Hg/m^3 in air (Roels *et al.*, 1987). The workers were tested for pulse rate variation in normal and deep breathing, the Valsalva maneuver, vertical tilt, and blood pressure responses during standing and isometric work. The only significant difference in subjective symptoms was an increased reporting of palpitations in exposed workers. The objective tests demonstrated an increase in pulse rate variations at 30 μg Hg/m^3 (extrapolated from blood level based on methods of Roels *et al.* (1987), which is indicative of autonomic reflex dysfunction.

Piikivi and Hanninen (1989) studied subjective symptoms and psychological performance on a computer-administered test battery in 60 chloralkali workers. The workers were exposed to approximately 0.025 mg/m^3 mercury vapor for a mean of 13.7 years. The vapor concentration was extrapolated from blood mercury levels based on the conversion factor in Roels *et al.* (1987). A statistically significant increase in subjective symptoms of sleep disturbance, and memory disturbance was noted in the exposed workers, although there was no difference in objective measures of motor, memory, or learning abilities.

A more recent study by Ngim *et al.* (1992) assessed neurobehavorial performance in a cross-sectional study of 98 dentists exposed to a TWA concentration of 14 μg Hg/m^3 (range 0.7 to 42 $\mu g/m^3$) compared to 54 controls with no history of occupational exposure to mercury. Exposed dentists were adequately matched to the control group for age, amount of fish consumption, and number of amalgam fillings. Air concentrations were measured with personal sampling badges over typical working hours (8-10 hours/day) and converted to a TWA. Blood samples were also taken (average 9.8 μg/L). The average concentration in air was estimated at 23 μg Hg/m^3 when the methods of Roels *et al.* (1987) were used. The average duration in this study of dentists was only 5.5 years, shorter than the above studies. The performance of the dentists was significantly worse than controls on a number of neurobehavorial tests measuring motor speed (finger tapping), visual scanning, visuomotor coordination and concentration, visual memory, and visuomotor coordination speed. These neurobehavorial changes are consistent with central and peripheral neurotoxicity commonly observed in cases of chronic mercury toxicity.

Liang *et al.* (1993) investigated workers in a fluorescent lamp factory with a computer-administered neurobehavioral evaluation system and a mood-inventory profile. The cohort consisted of 88 individuals (19 females and 69 males) exposed for at least 2 years prior to the study. Exposure was monitored with area samplers and ranged from 8 to 85 μg Hg/m^3 across worksites. The average level of exposure was estimated at 33 μg Hg/m^3 and the average duration of exposure was estimated at 15.8 years. The exposed cohort performed significantly worse than the controls on tests of finger tapping, mental arithmetic, two digit searches, switching attention, and visual reaction time. The effect of performance persisted after controlling for chronological age as a confounding factor.

V. Effects of Exposure to Animals

In laboratory animals mercury exposure resulted in adverse neurological and behavioral changes. Rabbits exposed to 28.8 mg/m^3 mercury vapor for 1 to 13 weeks exhibited unspecified pathological changes, marked cellular degeneration, and necrosis in the brain (Ashe *et al.*, 1953). Rats exhibited a decline in conditioned avoidance response with exposure to 3 mg/m^3 mercury vapor for 12 to 42 weeks. No histopathological changes were evident (Kishi *et al.*, 1978).

Congested lungs were observed in rats exposed to 1 mg/m^3 mercury vapor for 6 weeks, 100 hours/week (Gage, 1961). Rats exposed intermittently to 3 mg/m^3 mercury vapor for 12 to 42 weeks for 3 hours/day showed no changes in the respiratory system.

Rats exposed intermittently to 2.5 mg/m^3 mercury vapor for 21 days demonstrated prolongation of the estrous cycle and a decrease in the number of living fetuses (Baranski and Szymezyk, 1973), however, no differences in developmental abnormalities were observed.

VI. Derivation of Reference Exposure Levels

Derivation of Chronic Reference Exposure Level

Study	Piikivi and Hanninen (1989); Fawer *et al*. (1983); Piikivi and Tolonen (1989); Piikivi (1989); Ngim *et al*. (1992); supported by Liang *et al*. (1993)
Study population	Humans
Exposure method	Inhalation of workplace air
Critical Effects	Neurotoxicity as measured by :intention tremor; memory and sleep disturbances; decreased performance on neurobehavioral tests (finger tapping, visual scan, visuomotor coordination, visual memory); decreased EEG activity
LOAEL	25 μg/m^3 (3 ppb)
NOAEL	Not observed
Exposure continuity	8 hours per day (10 m^3/workday), 5 days/week
Average experimental exposure	8.9 μg/m^3 for LOAEL group
Human equivalent concentration	8.9 μg/m^3
Exposure duration	13.7 to 15.6 year average
Subchronic uncertainty factor	1
LOAEL uncertainty factor	10
Interspecies uncertainty factor	1
Intraspecies uncertainty factor	10
Cumulative uncertainty factor	100
Inhalation reference exposure level	0.09 μg/m^3 (0.0 1 ppb)

The USEPA (1995) based its RfC of 0.3 μg/m^3 on the same study but used an intraspecies uncertainty factor of 3, a LOAEL uncertainty factor of 3 and included a Modifying Factor (MF) of 3 for database deficiencies (lack of developmental and reproductive toxicity data). The criteria for use of modifying factors are not well specified by U.S. EPA. Such modifying factors were not used by OEHHA

The studies chosen to calculate the chronic REL all point to a LOAEL of approximately 0.025 mg/m3. When adjusted for worker ventilation and workweek exposure, the LOAEL becomes 0.009 mg/m3.

It is noteworthy that none of the above studies discussed in sufficient detail a dose-response relationship between mercury vapor inhalation and the toxic effects measured. Because none of the studies mention a level below which toxic effects were not seen after evaluation (a NOAEL), the extrapolation from a LOAEL to a NOAEL should be regarded with caution. Secondly, one study (Ngim *et al.*, 1992) demonstrated neurotoxic effects from mercury inhalation at an exposure level slightly above the other studies, but for a shorter duration. It is possible that mercury could cause neurotoxic effects after a shorter exposure period than that used in derivation of the chronic REL.

VII. Data Strengths and Limitations for Development of the REL

The strengths of the inhalation REL include the use of human exposure data from workers exposed over a period of years in a number of studies and occupations. The studies were consistent in terms of finding a LOAEL at about 0.025 mg/m^3. Major areas of uncertainty are the lack of observation of a NOAEL, the uncertainty in estimating exposure and the potential variability in exposure concentration, and the lack of reproductive and developmental toxicity studies.

In addition to being inhaled, airborne mercury can settle onto crops and soil and enter the body by ingestion. Thus, an oral chronic reference exposure level is also required for assessing risks from stationary sources in the Air Toxics Hot Spots program.

Derivation of U.S. EPA Reference Dose (RfD)

Study	U.S. EPA, 1987
Study population	Brown Norway rats
Exposure method	feeding and subcutaneous application
Critical effects	autoimmune effects in kidney
LOAEL	0.226 mg/kg-day (feeding); 0.317 mg/kg-day (subcutaneous)
NOAEL	none
Exposure continuity	
Exposure duration	up to 60 days
Average experimental exposure	
LOAEL uncertainty factor	10

Subchronic uncertainty factor	10
Interspecies uncertainty factor	10
Intraspecies factor	(The intraspecies and interspecies factors were combined into one factor of 10 to avoid an exceedingly large uncertainty factor.)
Cumulative uncertainty factor	1000
Oral reference exposure level	0.0003 mg/kg-day

Factors and Assumptions -- Dose conversions in the three studies employed a 0.739 factor for $HgCl_2$ to Hg^{2+}, a 100% factor for subcutaneous (s.c.) to oral route of exposure, and a time-weighted average for days/week of dosing. This RfD is based on calculations from a Drinking Water Equivalent Level (DWEL), recommended to and subsequently adopted by the U.S. EPA, of 0.010 mg/L: (RfD = 0.010 mg/L x 2 L/day/70 kg bw = 0.0003 mg/kg bw/day). The LOAEL exposure levels, used in the three studies selected as the basis of the recommended DWEL, are from Druet *et al.* (1978), Bernaudin *et al.* (1981) and Andres (1984), respectively.

The oral Reference Exposure Level for mercuric chloride is the U.S. EPA's RfD (IRIS, 1996). The principal study used was U.S. EPA (1987). A panel of mercury experts met at a Peer Review Workshop on Mercury Issues in Cincinnati, Ohio, on October 26-27, 1987, and reviewed outstanding issues concerning the health effects and risk assessment of inorganic mercury. The following five consensus conclusions and recommendations were agreed to as a result of this workshop: 1) The most sensitive adverse effect for mercury risk assessment is formation of mercuric-mercury-induced autoimmune glomerulonephritis. The production and deposition of IgG antibodies to the glomerular basement membrane can be considered the first step in the formation of this mercuric-mercury-induced autoimmune glomerulonephritis. 2) The Brown Norway rat should be used for mercury risk assessment. The Brown Norway rat is a good test species for the study of Hg^{2+}-induced autoimmune glomerulonephritis. The Brown Norway rat is not unique in this regard (since this effect has also been observed in rabbits). 3) The Brown Norway rat is a good surrogate for the study of mercury-induced kidney damage in sensitive humans. For this reason, the uncertainty factor used to calculate criteria and health advisories (based on risk assessments using the Brown Norway rat) should be reduced by 10-fold. 4) Hg^{2+} absorption values of 7% from the oral route and 100% from the s.c. route should be used to calculate criteria and health advisories. 5) A DWEL of 0.010 mg/L was recommended based on the weight of evidence from the studies using Brown Norway rats and limited human tissue data.

Three studies using the Brown Norway rat as the test strain were chosen from a larger selection of studies as the basis for the panel's recommendation of 0.010 mg/L as the DWEL for inorganic mercury. The three studies are presented below for the sake of completeness. It must be kept in mind, however, that the recommended DWEL of 0.010 mg/L and back calculated oral RfD of 0.0003 mg/kg-day were arrived at from an intensive review and workshop discussions of the entire inorganic mercury data base, not just from one study. In the Druet *et al.* (1978) study, the duration of exposure was 8-12 weeks; s.c. injection was used instead of oral exposure. In this study the development of kidney disease was evaluated. In the first phase the rats developed anti-GBM antibodies. During the second phase, which is observed after 2-3 months, the patterns of fixation of antisera changed from linear to granular as the disease progressed. The immune response was accompanied by proteinuria and in some cases by a nephrotic syndrome. Both male and female Brown Norway rats 7-9 weeks of age were divided into groups of 6-20 animals each. The numbers of each sex were not stated. The animals received s.c. injections of mercuric chloride ($HgCl_2$) 3 times weekly for 8 weeks, with doses of 0, 100, 250, 500, 1000 and 2000 µg/kg. An additional group was injected with a 50 µg/kg dose for 12 weeks. Antibody formation was measured by the use of kidney cryostat sections stained with a fluoresceinated sheep anti-rat IgG antiserum. Urinary protein was assessed by the biuret method (Druet *et al.*, 1978). Tubular lesions were observed at the higher dose levels. Proteinuria was reported at doses of 100 µg/kg and above, but not at 50 µg/kg. Proteinuria was considered a highly deleterious effect, given that affected animals developed hypoalbuminemia and many died. Fixation of IgG antiserum was detected in all groups except controls (Druet *et al.*, 1978). Bernaudin *et al.* (1981) reported that mercurials administered by inhalation or ingestion to Brown Norway rats developed a systemic autoimmune disease. The $HgCl_2$ ingestion portion of the study involved the forcible feeding of either 0 or 3000 µg/kg-week of $HgCl_2$ to male and female Brown Norway rats for up to 60 days. No abnormalities were reported using standard histological techniques in either experimental or control rats. Immunofluorescence histology revealed that 80% (4/5) of the mercuric-exposed rats were observed with a linear IgG deposition in the glomeruli after 15 days of exposure. After 60 days of $HgCl_2$ exposure, 100% (5/5) of the rats had a mixed linear and granular pattern of IgG deposition in the glomeruli and granular IgG deposition in the arteries. Weak proteinuria was observed in 60% (3/5) of the rats fed $HgCl_2$ for 60 days. The control rats were observed to have no deposition of IgG in the glomeruli or arteries as well as normal urine protein concentrations. Andres (1984) administered $HgCl_2$ (3 mg/kg in 1 mL of water) by gavage to five Brown Norway rats and two Lewis rats twice a week for 60 days. A sixth Brown Norway rat was given only 1 mL of water by gavage twice a week for 60 days. All rats had free access to tap water and pellet food. After 2-3 weeks of exposure, the Brown Norway $HgCl_2$-treated rats started to lose weight and hair. Two of the $HgCl_2$-treated Brown Norway rats died 30-40 days after beginning the study. No rats were observed to develop detectable proteinuria during the 60-day study. The kidneys appeared normal in all animals when evaluated using standard histological techniques, but examination by immunofluorescence showed deposits of IgG present in the renal glomeruli of only the mercuric-treated Brown Norway rats. The Brown Norway treated rats were also observed with mercury-induced morphological lesions of the ileum and colon with abnormal deposits of IgA in the basement membranes of the intestinal glands and of IgG in the basement membranes of the lamina propria. All observations in the Lewis rats and the control Brown Norway rat appeared normal.

The U.S. EPA reported its confidence in the RfD as: Data Base - High and RfD - High. No one study was found adequate for deriving an oral RfD; however, based on the weight of evidence from the studies using Brown Norway rats and the entirety of the mercuric mercury data base, an oral RfD of high confidence was derived. OEHHA believes that the RfD is an acceptable oral REL until better data become available.

VIII. References

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